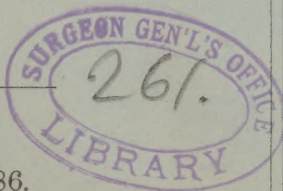


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TETANUS.

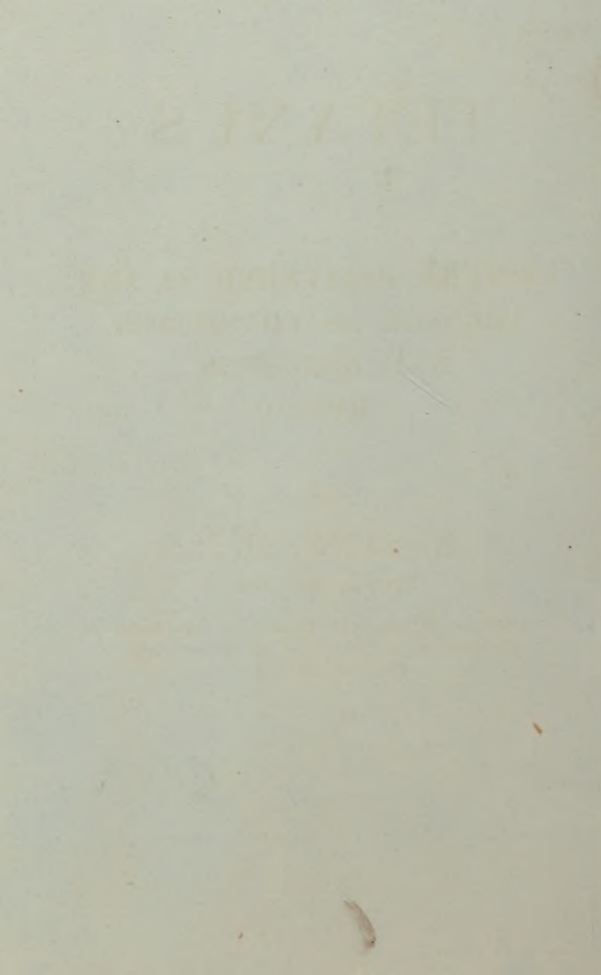
LECTURE DELIVERED AT THE
COLLEGE OF PHYSICIANS
AND SURGEONS,
CHICAGO.

BY
N. SENN, M. D.,
MILWAUKEE, WIS.,

SURGEON TO MILWAUKEE HOSPITAL, PROFESSOR OF
PRINCIPLES AND PRACTICE OF SURGERY AND
CLINICAL SURGERY.



1886.



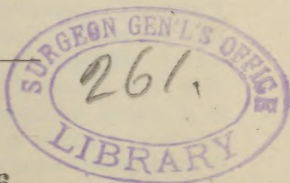
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TETANUS.

GENTLEMEN: Having finished the subject of wound infective disease proper, I shall call your attention this evening to a more infrequent wound complication, in which the symptoms point towards the cerebro-spinal centres as the primary and principal seat of the disease. This disease constitutes one of the most terrible in the long list of surgical affections, and up to the present time is wrapped in a great deal of obscurity. I refer to tetanus. Terrible, from the great mortality attending it, and from the intense suffering with which it is accompanied; obscure, because from the present standpoint of pathology, we are still in ignorance as far as its essential nature is concerned. Tetanus, I will define as being a disease of the nervous system, due to infection of a specific character, combined in many instances with peripheral irritation of a sensitive nerve; it is characterized by spasm of definite muscular groups, and attended by a continued form of fever. If I include the adjective "infective"

in tetanus, it is more on account of reasoning by analogy than positive pathological or clinical demonstration; nevertheless analogy should teach us that the well-marked period of incubation between the supposed time of infection and the development of the active symptoms resemble other forms of infective diseases very closely. That the direct cause of tetanus is due to a specific microbe, is claimed by a number of the most eminent writers, and further study and research will undoubtedly substantiate its infectious character. This disease is not a new one, inasmuch as it was well known to and described by Hippocrates, who recognized both an idiopathic and traumatic form, the latter of which he referred to wounds, abrasions, and to various pathological conditions independently of traumatism. Most all of the old classical works contain lengthy chapters on this subject, which reflect the most diverse opinions concerning its nature and etiology, which have been promulgated by different authors at various times.

In considering its geographical distribution, it has been observed that it is more frequently met with in the torrid zone, and more especially in India and South America. Statistics also show that in these countries the disease is noted for the gravity of its

symptoms and its greater mortality. In speaking of race influence, it can be stated that the colored races, as a rule, are more subject to it, and when suffering from the disease, it is usually attended by a greater mortality. Age appears to exert a predisposing influence, as after exclusion of tetanus neonatorum, at least 40 per cent. have occurred in patients 10 to 30 years of age. In order to prepare you for a pathological description of this affection, I shall call your attention to a form of toxic tetanus produced by the introduction into the circulation of certain chemical poisons. There is, however, this difference between this form of tetanus and traumatic tetanus; that in the former case the active symptoms of the disease make their appearance immediately after the poison has been brought in contact with the cerebro-spinal centres by the circulating blood. There is no period of incubation; no premonitory stage. The tetanic symptoms are well marked and fully developed as soon as the toxic effects of the drug have manifested themselves. The agents in our materia medica which are known to produce certain forms of tetanus, are strychnine, brucin, ergotine, and thebain; the forms of tetanic convulsions, however, vary according to the drug which has been ad-

ministered. Taking strychnine poisoning as a type of toxic tetanus, we find that the pathological conditions are always referable to vascular changes in the brain and spinal cord—changes marked by an increased afflux of blood, by an active hyperæmia or congestion, which later result in morphological changes indicative of the first symptoms of inflammation. Experiment has established the fact that the reflex action attending toxic tetanus is the result of excessive innervation from the spinal cord. If the spinal cord in an animal is crushed at a certain point, tetanic convulsions will only take place in the muscles supplied by the nerves from the intact portion of the cord. If, again, you should divide the posterior roots of the spinal cord near their exit from the spinal canal, or the posterior columns of the cord, there will be no response to the toxic irritation in the muscles supplied by the nerves which have been divided, or below the section of the cord. Furthermore, if previous to the intoxication you should remove the brain, the pons, or even the medulla oblongata, the toxic effect of the drug will not be impaired, showing conclusively that the tetanic convulsions or spasms are the direct result from the toxic effect of the drug upon the spinal cord.

In considering tetanus as it presents itself to the physician and surgeon, we still recognize an idiopathic and traumatic form. By the term "idiopathic," I mean a tetanus unattended or preceded by an appreciable traumatism. Traumatic tetanus, on the other hand, as its name indicates, is that which follows an injury. We place great stress upon the fact that there must have been somewhere a loss of continuity of surface, *an infection atrium*, as we term it, through which the specific germs gained entrance into the circulation. In many cases of so-called idiopathic tetanus, the traumatic lesion may have been so slight as to elude detection at the time when the disease made its appearance, and yet the traumatism may have been the direct and only cause of the disease. Of the seven hundred cases collected by Thamhayn, the disease had a well-defined traumatic origin in six hundred cases, while in the remaining one hundred cases there was no apparent evidence of previous injury found, and they were consequently classified as belonging to the spontaneous variety. To impress upon you the necessity of looking carefully for the source of infection in doubtful cases, I will briefly refer to a case that recently came under my observation, where from the history of the

case I thought I was dealing with a genuine case of idiopathic tetanus. The patient was a child five or six years of age, who had been ill for several days when it came under my care. The case presented all the symptoms of a mild type of tetanus, and in the absence of traumatism as a cause, I considered it as a typical case of so-called idiopathic tetanus. I had trismus, opisthotonos, and a mild form of continued fever with considerable disturbance in the function of the vascular system. After treating the case for a week or so, I accidentally one day found an exceedingly tender point in the sole of the right foot, which corresponded to a small cicatrix. On questioning more closely, the patient finally admitted having stepped upon a nail some time before, an accident which had been entirely overlooked by the parents and forgotten by the patient. Unless by accident the primary cause had been detected, this case would have been considered as belonging to the idiopathic variety, when in fact it was a well marked but mild case of traumatic tetanus. I simply make this allusion to put you on your guard in cases of so-called idiopathic tetanus, to search diligently and unceasingly for a tangible primary cause, as a slight injury or obscure cause of peripheral irritation might

be easily overlooked, which would lead to an incorrect interpretation of the etiology of the disease. There may be such a thing as idiopathic tetanus, without any apparent breach of surface, as specific germs may possibly gain entrance into the circulation through an intact mucous membrane in the same manner as in other instances of wound infective diseases, the microbes finding in the cerebro-spinal centres a favorable place of localization and reproduction. Future research, however, will have to verify or contradict this supposition.

In considering the exciting causes of tetanus, I will allude first to peripheral irritation. Before the infective nature of tetanus was recognized, it was generally considered that the disease was invariably the direct result of peripheral irritation. Clinical experience has demonstrated the important bearing of irritation of sensitive nerves, at least as a determining cause, inasmuch as in 380 cases reported by Thamhayn, it was found that 75 per cent. of them were the result of injury to the fingers, toes, hands, and feet—localities where we have an abundant supply of sensitive nerve filaments, so that it may be accepted as a well known and established fact that injuries of the hands and feet are more prone to be fol-

lowed by tetanus than wounds in any other locality.

Another form of peripheral irritation, aside from the traumatic, are pathological conditions due to inflammation, which affect the sensitive nerves the same as when the irritation is produced by an injury. Tetanus produced in this manner has been observed as a complication in pleuritis, pneumonia, metritis; in fact, as a result of pathological conditions which involve in the same manner an extensive area of sensitive nerves as the effects of traumatism. In the idiopathic form of tetanus, authors still recognize a central lesion or point of irritation independently of any peripheral or eccentric pathological irritation. By this I mean central irritation by pathological conditions involving directly the brain or spinal cord, independent of traumatism. Cases have been observed in the post-mortem room where well marked pathological conditions existed in the brain and spinal cord, independent of either traumatic pathological peripheral irritation, where during life the characteristic symptoms were well marked. These cases may serve to explain the theory that the specific germs of tetanus once introduced into the system may have a special elective affinity for the nerve centres, and may there manifest their toxic

influence by producing pathological conditions which induce tetanic spasms. Specific infection, then, must be recognized as the most important and essential factor in the etiology of tetanus. Recent literature on this subject seems to have established the fact, that like wound infective diseases, tetanus is simply an expression of a specific form of intoxication, which implicates, principally, the cerebro-spinal centres, the result of the introduction of specific microbes. This hypothesis does not lack analogy in diseases which are marked by a well-defined period of incubation, where a certain time elapses between the introduction of the virus and the active development of the symptoms; in other words, diseases preceded by a period of incubation, in contradistinction to diseases which are caused by the introduction of a formed chemical poison, means simply that the germs introduced at the time when the injury was inflicted keep on multiplying until a sufficient number of them have been generated in the system, when the toxic effects are announced by the active development of symptoms. If tetanus were the result of the introduction into the system of a pre-formed or chemical poison, the active stage of the disease would commence as soon as the poison had entered the circulation,

which evidently has never been the case. If, however, a certain period elapses from the time of supposed infection until the active development of the disease, we recognize the fact by well demonstrated analogy that these specific germs have been introduced into the system, and have found a favorable soil for their growth and multiplication in the central nervous system until their direct toxic effects are manifested by tetanic convulsions. The period of incubation in tetanus is not as well marked and definite as in some other infectious diseases, but the same can be said of other affections where there can be no doubt concerning their microbial origin, hydrophobia, for instance. The most characteristic symptom of tetanus consists in tonic spasm of certain well-defined muscular groups, as a rule, the affection following in a descending direction. We will, for the sake of convenience, assume that tetanus has followed a compound comminuted fracture of the leg, and will describe the symptoms in their proper order. About the fifth or sixth day (the earliest period of incubation) our patient will probably complain of a certain ill-feeling in the broken limb, marked by irregular jerking; if, at the same time, he complains of general malaise, loss of appetite, of symptoms indicative of infection, we

should realize that these premonitory symptoms are but the precursors of the gravest of all wound complications—tetanus. If these early symptoms are followed a little later by the appearance of trismus, there can be no further doubt as to the nature of the disease. By trismus, I mean a contraction of the muscles of mastication, as evidenced by difficulty in opening the mouth. Going still further down, other muscular groups are implicated, and we have what is known as opisthotonos, a bending backwards of the body in contradistinction to emprosthotonos, a bending of the body in the opposite direction. If the muscles on one side of the chest are affected, we have pleurosthotonos; if still further, all the muscles in the body (usually the extensors, however,) are affected, and the entire body becomes rigid, we have orthotonos. If the muscular spasms have become well developed, involving the area of the respiratory muscles, we expect an impairment of the function of respiration. By spasmodic contraction of the muscles concerned in that function, respiration becomes imperfect on account of the constant rigidity of the muscles which are active in producing the respiratory movements.

The temperature is increased in almost every case, the rise depending upon the in-

tensity of the infection, the acuity of the attack, and especially the rapidity with which the disease progresses. A limited area of muscular rigidity and a slight rise in temperature, are favorable prognostic indications. In some instances the temperature has been found unusually high. A temperature of 108° , even 110° , as observed by Wunderlich and Billroth, denotes great danger, and is almost without exception followed by a fatal termination. These authors have placed stress upon the importance of temperature in tetanus, watching its rise in the axilla at different times of the day, and observing that an increase of temperature beyond 104° or 105° is usually followed by death. Another curious fact has been noticed, and that is an increase in temperature after death. Wunderlich explains the high temperature in tetanus by assuming a loss of function, or a loss of control of the heat moderators in the brain, the heat centres being impaired by the direct action of the poison upon this particular portion of the cerebral mass. The post-mortal rise in temperature has been attributed by Fick and Huppert to evolution of heat during the coagulation of the myosin. The sensorium usually remains unimpaired to the last, showing distinctly that that portion of the brain concerned in that function remains un-

impaired, that the disease for some unknown reason expends itself upon the motor tracts, and especially upon that portion of the cerebro-spinal system connecting the brain and spinal cord, the medulla oblongata. When the spasms are severe and prolonged, the respiration becomes impaired, the pulse small and rapid, and the surface of the body is bathed with a profuse perspiration. As the disease progresses, evidences of capillary stasis become more and more apparent, the eyes are suffused, and the visible mucous membranes livid; the tongue is coated, appetite diminished, and the bowels are obstinately constipated; all causes of peripheral irritation, as a draft of air, loud noises, etc., will provoke easily a repetition of spasmodic attacks.

The first attempt at an intelligent explanation of the morbid anatomy of tetanus was offered by Rokitansky, who found on examining the nerves supplying the injured part evidences of inflammation in the nerve itself, and hence described the disease as a form of neuritis, a *neuritis ascendens*. As proof of this view of the disease he described the pathological conditions found in the affected nerves as consisting in an exudation between the nerve fibres, granular degeneration and hyaline changes in the nerve fibres themselves, finally resulting in com

plete disorganization and disintegration. He regarded the disease essentially as a "neuritis ascendens," the morbid process commencing in the nerve at the point of injury, and extending by continuity to the cerebro-spinal centres. All pathologists agree that the brain and spinal cord are always found in a state of congestion, but some go still further—Lockhart, Clarke, and Dickinson, for instance, asserted that the brain was not only hyperæmic, but that there was an actual inflammation of the cortex of the brain itself, resulting in granular disintegration. Others have found, on examining the spinal cord, pathological changes well marked in that portion of the nervous system, consisting of hyperæmia, extravasation, softening, increased proliferation of connective tissue, degeneration of the anterior and posterior columns. Tyson, on the other hand, found in two cases well marked degeneration of the central canal and disintegration of the posterior columns of the cord. Aufrecht found cellular atrophy in the anterior and posterior cornua of the cord and granular degeneration of the gray substance of the cord. From all that I have stated it appears evident that so far no unanimity exists among pathologists in localizing the central lesion, but from what we have learned it becomes

apparent that the most constant pathological changes are found in the upper portion of the spinal cord.

In considering the diagnosis, particular attention should be paid to the history of the case, fully eliciting the possibility of any previous traumatism or infection serving as a tangible cause. In cases of spasm of definite groups of muscles, we should ascertain the existence or absence of fever by the use of the thermometer.

Although no uniform pathological condition appears to have been found characteristic of tetanus, the weight of evidence points towards the spinal cord as the central seat of the lesion, in this respect showing a resemblance to hydrophobia, an affection which it simulates in many respects.

Muscular spasm resulting from a neurotic source and limited to a group of muscles unattended by general infection, is not marked by rise in temperature. If, on the other hand, we have muscular spasm attended by an increase of temperature (particularly if we have reason to believe that it is the result of previous traumatism or infection), the case becomes suspicious, and merits a scrutinizing examination. In tetanus, as a rule, the muscles on the extensor side are affected, while in cases of inflammatory dis-

ease in bones and joints, the contractions occur on the flexor side.

In the differential diagnosis, consider the possibility of toxic tetanus from poisoning with strychnine or any of that class of remedies which are known to produce tetanic spasms. The mental condition of the patient and the surrounding circumstances of the case will aid you greatly in eliminating such a cause. In strychnine poisoning, orthotonos is produced as soon as the drug has been absorbed, and tetanic spasms of muscular groups pass in a descending direction.

Hysteria may simulate tetanus, but you must remember that your hysterical patient cannot, for any length of time, maintain muscular spasm limited to definite muscular groups. The pupils of the eyes in tetanus are generally contracted, and the temperature is increased, while in hysteria both of these symptoms are absent.

The next disease which may be mistaken for tetanus is cerebro-spinal meningitis. If an epidemic of this disease prevails in your locality, carefully consider the differential diagnosis. In cerebro-spinal meningitis the symptoms point equally to an affection of the base of the brain, while brain symptoms proper do not belong to the clinical history

of tetanus; hence, you will encounter symptoms in the former which point towards an active inflammation of the meninges of the brain, as well as of the spinal cord. If the disease has lasted for a sufficient length of time, you will look for the symptoms aside of muscular spasm which characterize cerebro-spinal meningitis. Basilar meningitis is attended by symptoms indicating irritation at the base of the brain; and as it is almost always of a tubercular nature, you will carefully inquire into the history of the case, and concomitant tubercular lesions.

The last disease which it might be mistaken for is hydrophobia. The muscular spasms in hydrophobia are more of a clonic character, and remain limited to the muscles of deglutition and respiration. In the prognosis you must be guided, in the first place, by the extent of the central lesion, as evidenced by the number of muscular groups affected. If the central irritation is circumscribed, only a limited number of muscular groups are affected, and when this is the case, we are usually dealing with a mild form of the disease. If, on the other hand, in the early history of the disease there is rigidity of an extensive muscular area, the disease is a grave one; and more particularly if at the same time, as will likely be the case, there is a high temperature. Time is one of your

most important elements in rendering a prognosis. Hippocrates always considered the prognosis dubious the first four days. Severe cases may terminate fatally from the second to the sixth day, usually, so that time gained after the sixth day, with the symptoms remaining stationary, the prognosis becomes more favorable as time increases. In the mild cases of tetanus, terminating in recovery, it may take weeks and sometimes months before all muscular rigidity has disappeared. I have observed several severe and acute cases where within forty-eight hours after the commencement of the first symptoms, death occurred; but after the sixth day, with symptoms remaining stationary, the surgeon's hope increases as time elapses.

Another important point is the probable cause of the disease. If the injury has been slight, infection limited, the disease assuming a mild type, your prognosis may be favorable; but should the symptoms develop themselves incidental to injuries grave in themselves, the prognosis becomes correspondingly serious, so that in tetanus attending severe lesions, followed by traumatic infection of other types, attended, perhaps, by septicæmia or pyæmia, our prognosis always must be unfavorable; in other words, if there is no possibility of removing the primary cause or

source of infection or irritation, the prognosis increases in gravity.

Like in all other forms of wound infective diseases, the prophylactic treatment is the most important. Practically, it is important to recognize the microbic origin, and institute early and efficient treatment in accordance with this supposition. I therefore, again, emphasize the necessity of adopting aseptic measures in the treatment of wounds, with a view not only of preventing suppuration, but also other forms of infection. There can be no question but that since the introduction of antiseptic surgery tetanus has been uncommon. The clinical fact is patent that it is prone to follow injuries where a foreign body has remained in the wound, and where subsequently from this cause there is greater danger of infection on the one hand, and peripheral irritation on the other; this is especially true of injuries about the hand. It is therefore of paramount importance to treat slight injuries of the hand, and more especially 4th of July injuries, which so frequently end in tetanus, with the greatest care. No matter how trifling the injury may be, disregard in this direction has only too often been followed by the most serious consequences. A small wound of the hand when neglected may serve

as an avenue for the ingress of germs, which according to their action may destroy life in a variety of ways. If necessary, enlarge the wound, search carefully for foreign bodies, if there is reason to believe that they are present; take plenty of time to secure an aseptic condition for the wound, and conduct the subsequent treatment on strictly antiseptic principles. If suppuration should take place, be sure to secure efficient drainage, and resort to frequent antiseptic irrigations. By following these directions you will not only have the satisfaction of securing the most favorable condition for the healing of the wound, but you will have, at the same time, carried out the most efficient prophylactic treatment against tetanus.

In the curative treatment, we consider first the importance of removing the source of infection and irritation. If a foreign body remains in the wound, search diligently for it, and if possible remove it. If the injury is attended by inflammation, burrowing of pus, destruction of tissue underneath the skin, secure ample and efficient drainage; in other words, convert the wound, as near as you can, from a septic into an aseptic condition. If we are dealing with a painful or tender cicatrix, which by producing peripheral irritation may determine tetanic

spasms, it is proper to excise it. We can readily conceive that the infection may have been so mild as to produce only slight changes in the spinal cord, which but for some peripheral irritation would not have resulted in muscular spasms, and these are the cases where the removal of the exciting cause of the spasms is followed by a cure. I recollect a case of a mild form of tetanus, the result of a slight injury at the distal extremity of the index finger, which left a painful, tender scar, where the trismus was always increased by pressure, and which yielded promptly to removal of the exciting cause.

Another practical point in the treatment is the consideration of the propriety of amputation. If we are dealing with a compound comminuted fracture of the leg or arm, attended by all the symptoms of wound infection, and if present indications point towards the fact that the limb, independently of the existence of this complication, could not be saved, it is only rational to resort to amputation. It is, however, proper to state that in the great majority of the cases thus treated the tetanus continued unabated after the operation, and only in exceptional cases has life been saved by this procedure. Recognizing the fact that according to Rokitan-

sky's theory, tetanus simply means "an ascending neuritis from the point of injury to the spinal cord," attempts have been made to arrest the progress of the disease by interrupting the nerve above the disease by nerve section, but results have shown that this operation had no effect in modifying the termination of the disease. You will readily understand *why*, if you assume its central location and its infective character. If tetanus were simply a "neuritis ascendens," we might reasonably expect to arrest the progress of the disease by division of the nerve trunk, by making the section through healthy tissue on the proximal side of the inflamed portion of the nerve. Granted that this operation would be efficient in preventing extension of the inflammatory process, it would not be applicable in all instances, from the inability of the surgeon to locate the disease with sufficient accuracy. Imagine an injury involving the palm of the hand, where we have filaments of different nerves implicated; it would be difficult if not impossible to ascertain in every case which nerve trunk was the seat of irritation. Taking it for granted that the neuritis is simply a local expression of the nerve injury, and that the real cause of tetanus consists in a specific infection, expending itself upon the

brain and spinal cord, all efforts at correcting or curing the disease by submitting nerve trunks to operative measures are in discord with the true pathology of the disease. A somewhat similar and more recent operation for the same object as nerve section, we find in nerve stretching. For the last few years it has become the operation for all obscure nervous diseases, such as locomotor ataxia and central irritation of various kinds; consequently it is not surprising that it has been resorted to in the treatment of tetanus.

Benedict collected twenty-four cases of traumatic tetanus treated by nerve-stretching, of which number four recovered. I have stated that the disease in the milder form shows a tendency to cure itself; consequently, all operative measures which have been resorted to for the purpose of arresting its progress must be accepted with a great deal of caution, inasmuch as statistics have shown, on the whole, that twenty-five per cent. have recovered without operative interference by the ordinary treatment; consequently, we have no proof that these four cases would not have recovered without nerve-stretching. All the possible good that can accrue from operations on nerve-trunks is simply to interrupt the nerve-current between the central

disease and the peripheral irritation, and in many instances it is impossible to decide which of the larger trunks connects the two.

The medical treatment consists in the administration of drugs which are known to relieve muscular spasm. In animals tetanized with strychnine the spasms are promptly relieved by injecting hydrate of chloral into the veins. Woorara has been given with a view of relieving muscular spasm. On account of the potency of this remedy, its effects must be carefully watched and its use promptly suspended as soon as its physiological action becomes apparent. It is best administered hypodermatically. Bromide of potassium in large doses is well known to cause cerebral anæmia, and on this account it should be given to relieve the hyperæmia which is always present in the cerebro-spinal centres. A combination best adapted to fulfil the two most urgent indications, to relieve cerebral and spinal congestion and to overcome muscular spasm, are chloral and bromide of potassium. The remedies should be given in large doses, frequently repeated, until the desired result has been obtained. In severe cases, chloroform should be administered by inhalation with a view of relieving urgent symptoms; cold drafts of air, noise, and all forms of peripheral irritation, should

be carefully excluded from the patient's room, for the purpose of securing rest, and with a view of not aggravating reflex spasm. Alcoholic stimulants and external heat are indicated when the heart's action has become feeble from general capillary stasis.

